

Pseudotumoral lesion: pathology and follow-up

Andres Villa¹ · Cecilia Quarracino¹ · Antonio Colobraro¹

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A 34-year-old woman arrived at our hospital's emergency department with 1 week of progressive and oppressive occipital headache, associated with photophobia, nausea and vomiting, and right-sided brachio-crural paresis.

At physical examination she had expressive aphasia and moderate right-sided hemiparesis. There were no particular findings in her basic blood exams. Magnetic resonance imaging (MRI) showed a hypointense T1 and hyperintense T2 left frontotemporoparietal expansive lesion with mass effect, partially collapsing the ipsilateral ventricle with a midline shift (Fig. 1a, b). Furthermore, MRI showed a perilesional brain edema, but no enhancement with gadolinium. There was no restriction of diffusion and no other lesions were present. Given the diagnostic impression of a neoplastic process, she was admitted as an inpatient and a whole body CT scan, broader laboratory tests, including an HIV serology, mammography and mammary echography were ordered.

In the following hours she evolved showing intracranial hypertension signs (hypertension, bradycardia, headache intensity increase) and was therefore started on intravenous dexamethasone 8 mg every 6 h. Due to persistent symptomatology a neurosurgical evaluation was conducted and the decision was made to perform an urgent neurosurgery, consisting in a decompressive craniectomy with biopsy. In the immediate postoperative period her motor deficit slowly began to improve.

Pathology studies showed reactive astrogliosis and demyelination with the presence of histiocytes, microvascular proliferation and a sparse lymphocytic component (Fig. 1c). Luxol fast blue revealed a decrease in myelinated fibers. Immunohistochemistry was positive for CD68 in macrophages, which led to the diagnosis of an active demyelinating lesion (Fig. 1d).

She was started on a daily dose of 60 mg of prednisone, which was slowly tapered in the following 6 months. She exhibited a complete clinical resolution of symptoms at the three-month follow-up, which persists to this day, 34 months after the clinical presentation.

Neuroimaging studies also improved; a follow-up MRI at the 24th month hardly exhibited any signs of the previous expansive lesion (Fig. 1e, f). In follow-up MRIs, she never developed any other demyelinating lesions (not shown).

Tumefactive demyelinating lesions (TDL) are inflammatory plaques larger than 2 cm in size [1–3]. Generally, most of these lesions enhance with gadolinium contrast with an incomplete ring and have a homogeneous centre in T2 sequences [1–4]. There also seems to be a predilection for the frontal and parietal lobes, although other locations have also been reported [2, 5]. In a 52 case-series, Atlintas et al. [3] found a male to female ratio of 1:2. Almost half of these patients had been previously diagnosed with multiple sclerosis, whilst it was the first inflammatory episode for the rest.

Our patient had a typical TDL localization but without the classic contrast enhancement. She did not have a previous diagnosis of inflammatory disease nor did she have any other prior brain lesions to suggest this. Although expansive lesions are commonly a consequence of a neoplastic or infectious disease process, we believe that the possibility of a demyelinating disease should be considered

✉ Andres Villa
andresmariavilla@gmail.com

¹ Hospital Ramos Mejía, Buenos Aires, Argentina

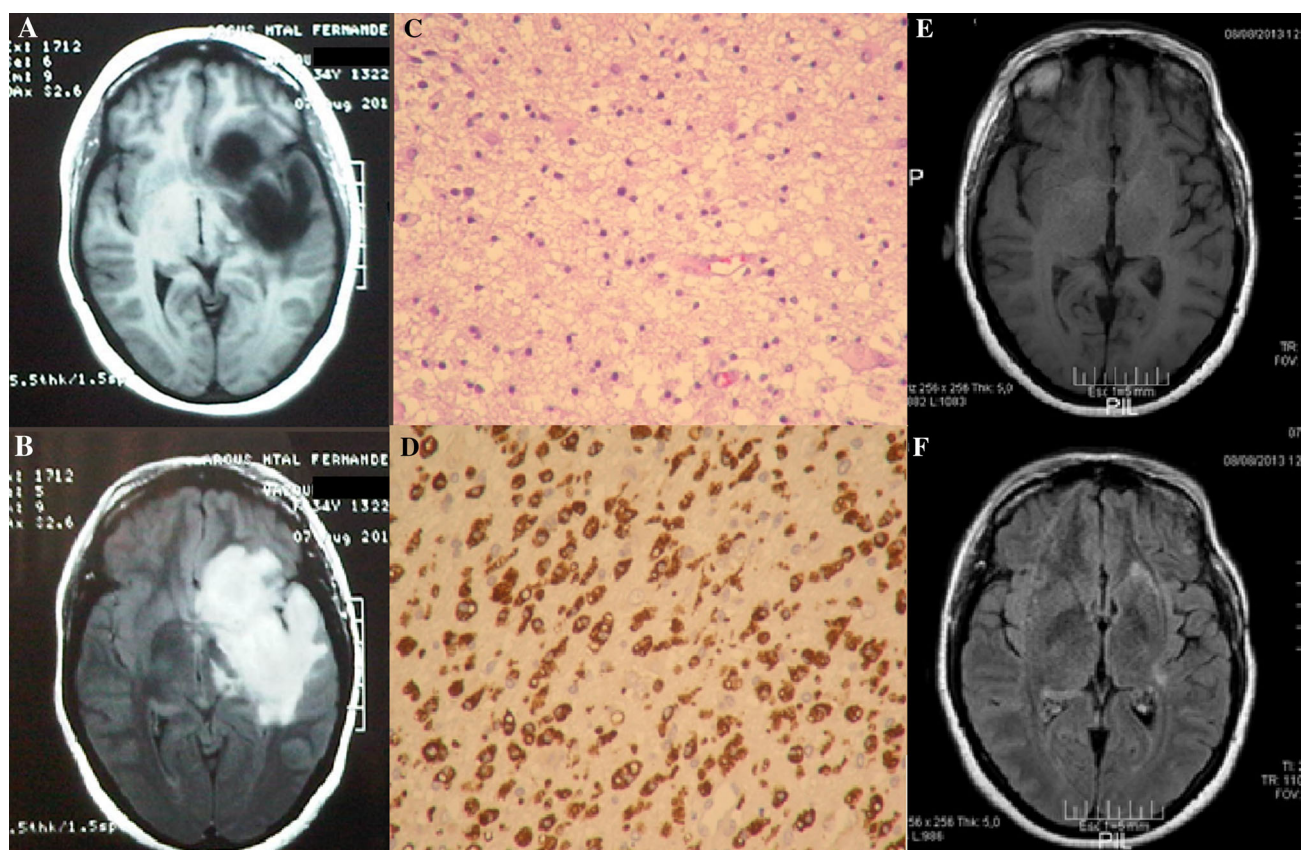


Fig. 1 **a, b** Brain MRI at diagnosis showing a hypointense T1 (**a**) and hyperintense FLAIR (**b**) fronto-temporal lesion with mass effect, without contrast enhancement (not shown). **c** Biopsy specimen with hematoxylin and eosin $\times 200$ white matter with a lymphocyte and

macrophage infiltrate. **d** Biopsy specimen with CD68 immunostaining $\times 200$ showing foamy immunoreactive macrophages. **e, f** Brain MRI at 30-month follow-up showing minimum residual signs of the previous lesion at T1 (**e**) and FLAIR (**f**) sequences

within the diagnostic algorithm in order to prevent invasive diagnostic surgery.

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Conflict of interest The authors declare that there are no conflicts of interest.

Ethical approval This study was approved by the Medical Ethics Committee of our center and conducted in accordance with the ethical standards recognized internationally.

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Informed consent Written consent was obtained from the patient prior to the manuscript.

References

1. Lucchinetti CF, Gavrilova RH, Metz I et al (2008) Clinical and radiographic spectrum of pathologically confirmed tumefactive multiple sclerosis. *Brain* 131:1759–1775
2. Hardy TA, Chataway J (2013) Tumefactive demyelination: an approach to diagnosis and management. *J Neurol Neurosurg Psychiatry* 84:1047–1053
3. Altintas A, Petek B, Isik N et al (2012) Clinical and radiological characteristics of tumefactive demyelinating lesions: follow-up study. *Mult Scler* 18:1448–1453
4. Bower RS, Burrus TM, Giannini C et al (2010) Teaching neuroimages: demyelinating disease mimicking butterfly high-grade glioma. *Neurology* 75(2):e4–e5
5. Rovira A (2014) Tumefactive idiopathic inflammatory demyelinating lesions: a diagnostic challenge. *Mult Scler* 20(5):634–635